traditions, in which is crystallized the wisdom of its past; and that seniors who have risen to eminence and who have that soundness of judgment which the young, however learned and able, rarely have, can best determine when it is and when it is not judicious to depart from those traditions.

And one word more: I envy you; not only because you are young and I am old, for the old always envy the young; but also, and chiefly because you are to enter upon the practice of the greatest of the professions; a profession whose members have it in their power to do more than other men in relieving human suffering and alleviating human misery; a profession whose history is a story of continued growth and progress, of brilliant discoveries in science and beneficent improvements in methods of treatment and of operation, and, in many instances, of martyrdom to duty.

I close with the expression of the hope that you will walk worthy of your high vocation, and that in the years to come you will be loyal to your Alma Mater and do honor to your profession and your College.

SOME OBSERVATIONS ON ANEURYSM AND ARTERIOSCLEROSIS.[1]

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HISTORY.

In tracing the history of the study of aneurysm, we find it divides itself into two periods. First, that in which the aneurysm was looked upon as a localized disease of the vessel; second, that in which arteriosclerosis was recognized, aneurysm being considered in association with an advanced stage of this change in the arterial wall.

The first of these periods, that of aneurysm, divides itself into two—that in which the peripheral aneurysm was recognized and treated, and that in which the deep seated, like the thoracic aneurysm, was recognized.

The steps in the development of our present knowledge of arterial disease as they are here presented will be appreciated by following these charts.

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[1] The Mütter Lecture, delivered before the College of Physicians of Philadelphia.

CHART I.

Historical Chart. (Chronological.)

- A. Epoch of Ancient Medicine (1500 B.C.-210 A.D.).
  - Egyptian Medicine.
  - Assyro-Babylonian Medicine (Ignorance and Superstition).
  - Hebrew Medicine (1500-400 B.C.).
  - Indian Medicine.
  - Greek Medicine (400-300 B.C.) } Aneurysm confused with other tumors.
    - Hippocrates (460-377 B.C.) }
    - Aristotle (384-323 B.C.) }
    - School of Alexandria (300 B.C.).
  - Roman Medicine (100 B.C.-210 A.D.).
    - Asclepiades (100 B.C.).
    - Celsus (First Century).
    - Galen (131-210 A.D.). (Term "Aneurysm"—"widening." Arteries contain blood, not air.)

The epoch of ancient medicine, which may be said to extend from 1500 B.C. to 200 A.D., is represented by Egyptian, Assyro-Babylonian, Hebrew, Indian, Greek, and Roman medicine. In this epoch, doubtless, peripheral aneurysms were seen but confused with tumors of other nature. In Greek and Roman medicine occur the names of Hippocrates (460–377 B.C.), Aristotle (384–323 B.C.), Herophilus and Erasistratus of the school of Alexandria (300 B.C.), Asclepiades (100 B.C.), Celsus (first century), Aretaeus (second century), and Galen (131–210 A.D.). It is the last-named who probably makes the first definite reference in literature to the condition and who used the term *'Ανέυρυσμα*, meaning a dilatation.

It will be remembered that it was Galen who declared that the arteries contained blood not air, the latter view having been held by Erasistratus of the school of Alexandria. (Dezeimeri's *Dictionnaire Historique de la Médecine Ancienne et Moderne.*) The translation of the passage from Galen's work in which he refers to aneurysm is the following: "When the mouth of the artery is opened the affection is called *'Ανέυρυσμα* (dilitation). The same thing happens when the artery being wounded, the incumbent skin is cicatrized, but the wound of the artery remains, being neither conglutinated, covered with a cicatrix, nor shut by incarnations. Affections of this kind are known by the pulsation of the arteries, or more especially by compressing the artery, for then all the tumor disappears, the matter that caused it recurring into the artery." This was written in the latter part of the second century. (Quoted by Robert James in *A Medical Dictionary*, 1743, under "Aneurysm.")

CHART II.

- Mediæval Medicine (300-1400 A.D.).
  - Byzantine Medicine.
  - Oribasius (326-403 A.D.). Recognized peripheral aneurysms.
  - Paul of Ægina (625-690 A.D.). Recommended ligation of brachial artery for aneurysm at bend of elbow.
  - Arabian Medicine.
    - Avicenna (980-1036 A.D.). Writes on peripheral aneurysm.

The next epoch, that of mediæval medicine, which may be said to extend from 200 to 1400 A.D., and which consists of the Byzantine (300–700) and Arabian (800–1000) periods, also the school of Salerno (1000–1200) and that of Mundino (Mundinus) (1276–1326), contains but indefinite reference to peripheral aneurysms, though in the Byzantine period, Oribasius (326–403) ligated arteries for cure of aneurysm, and Paul of Ægina (625–690) recommended ligation of the brachial artery a few fingers' breadth below the axilla for cure of aneurysm at the bend of the elbow (Hodgson), and in *Arabian Medicine* we find that Avicenna (980–1036) was familiar with peripheral aneurysms. Most authors attribute the first recognition of peripheral aneurysm to Avicenna, though from the above it is evident that these aneurysms were recognized and even treated four or five hundred years before Avicenna.

In these periods, however, all of those fundamental facts of gross anatomy and physiology were being sought for, so that beyond a few observations of historic interest these periods contain no contributions to our knowledge of arterial disease. Until anatomy was systematically studied, this whole subject remained in obscurity. The advances in anatomy, indeed, amounted to very little up to this time, the beginning of the epoch of modern medicine which begins with the time of Linacre (1461–1524) and extends to our own. Prior to this (1400) the studies of human anatomy may be estimated by the fact that Mundino (1276–1326), who was professor of anatomy at the University of Bologna during the latter part of the thirteenth century, "dissected in eleven years only three human bodies." From the period of the Alexandrian school (300 B.C.) to this time of Mundino (1300 A.D.) the human body had never been dissected (Chapman). With the beginning of the sixteenth century, peripheral aneurysms were well recognized, Paré (1517–1590) introducing the method of ligating on both sides of the aneurysm, allowing, however, the intervening area to slough out. He also definitely differentiated abscesses from aneurysms.

The discussion now turns to the possibility of aneurysms of deep-seated vessels. To Vesalius (1514–1565) is due the credit of making the first diagnosis of intrathoracic aneurysm during life. This aneurysm followed injury and was detected by examining the back of the patient; a point of great clinical importance rarely mentioned, and one alluded to in a recent paper by Osler.

"The Doctrine of Dilatation of the Aorta," however, made so slow a progress that in the year 1596 when Sylvaticus published his "Treatise on Aneurysm" he did not mention internal aneurysm, and many years afterward (1658) Riolanus asserted that "aneurysm happened but seldom in the trunk of aorta in consequence of the thickness of its parietes." Elsnerus prefixed to his observations concerning aneurysm of the arch of the aorta *De Paradoxico Aneurismate*. The *Sepulchretum Anatomicum* of Theophilus and the

Historia Anatomica Medica of Lieutaud, Morgagni characterized as "unsystematic and vague."

Of thoracic and deep-seated aneurysm Morgagni says: "From the year 1670 they ceased to be considered as rare occurrences; and we have now acquired such familiarity of the disease from its causes and symptoms that we believe it may be distinguished, even when very small, and entirely concealed within the thorax." Malpighi, who was partly a contemporary of Morgagni "accounted the knowledge of dilatation of the aorta among the improvements of (his) age."[1] About this time (1670) Valsalva (1666–1723) tied the carotid of dogs repeatedly, and in one instance he tied both carotids, the dog living twenty-two days (Hodgson).

Much valuable clinical and pathological information was added by Johannes Mariana Lancisi (1654–1720) in his *De Motu Cordis et Aneurismatibus*, and by Haller (1708–1777), who in his *Opuscula Pathologica* (vol. x. and book vii., p. 127) introduces the term "Atheroma" and demonstrates collateral circulation, also by John Hunter (1728–1793), who tied the distal end of the vessel for cure of aneurysm (1785).

In the writings of Malpighi (1628–1694), Valsalva (1666–1723), and Morgagni (1682–1771), as embodied in his *De Sedibus et Causis Morborum,* published in 1760, are systematic ante-mortem and post-mortem descriptions of aneurysm. Here we find accurate reports of aneurysm of the splenic, popliteal, and inguinal arteries. Of arteries in general we find reports on ulceration of the coats of the vessel. Of the aorta we find reports on atheromatous coats, thickening and induration, inflammation, opaque spots, ossification, callous, cartilaginous deposits, incipient aneurysm, advanced aneurysm, and ruptured aneurysm. Case 26 is the report of sudden death resulting from rupture of one of the coronary arteries.

In 1805 Sir Astley Cooper ligated the carotid artery, a step which, as already stated, had been taken by Valsalva experimenting upon dogs. In 1811 Joseph Hodgson contributed his *Treatise on the Disease of the Arteries* which contains observations upon the pathology and treatment. Antonio Scarpa (1748–1832) also published an exhaustive *Treatise on Pathology and Surgical Treatment of Aneurysm* which was translated into English from the Italian about 1830. The clinical contributions of Laennec (1781–1826) and of that great teacher Louis (1787–1872) must not be omitted. From Corvisart (1755–1821) we get the term "vegetation."

Peripheral aneurysms then were recognized and treated as far back as the third and fourth centuries. Deep-seated aneurysms were recognized about the end of the seventeenth century.

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[1] Seats and Causes of Disease, xviii., 14; Cook's translation, 1824, p. 330.

CHART III.

Historical Chart. (Chronological.)

Modern Medicine (1400–1900 A.D.).
Revival of Anatomy. Vesalius (1514–1565). Thoracic aneurysm diagnosed during life.
Practical Surgery. Paré (1517–1590). Ligates on both sides of aneurysm.
Circulation. Vascular system including capillaries demonstrated.
Harvey (1578–1657). Circulation.
Malpighi (1628–1694).
Van Leeuwenhoek (1632–1723). } Capillaries.
Gross. Pathology.
Morgagni (1682–1771)
Malpighi (1628–1694) } Peripheral and deep-seated aneurysm and other diseases
Valsalva (1666–1723) of the arterial wall recognized and described.
Medical, Surgical, and Pathological.
Lancisi (1654–1720). "De Motu Cordis et Aneurysmatibus."
Haller (1708–1777). Term "Atheroma."
Auenbrugger (1722–1809). Percussion.
John Hunter (1728–1793). Ties distal end for cure of aneurysm.
Astley Cooper. Ligates carotid (1805).
Scarpa (1748–1832). "Treatise on Pathology and Surgical Treatment of Aneurysm."
Joseph Hodgson. "Treatise on Pathology and Treatment of Aneurysm" (1811).
Corvisart (1755–1821). Term "Vegetation."
Laennec (1781–1826). Auscultation.
Abernethy. "Origin and Treatment of Aneurysm" (1817).

Coming more to our own times, we find the observations upon the diseased arteries more specific as indicated by such terms as "arteriosclerosis" introduced by Lobstein in 1834 and "endarteritis obliterans" by Friedländer. "Arteriocapillary fibrosis" was introduced by Gull and Sutton, who recognized the condition as an independent affection.

The first theory upon which arterial disease was studied was that of inflammation, a view held by such men as Scarpa, Haller, Kreysig, Lobstein, and Bizot. It must be remembered, however, that at the time of these observers no very clear view of the pathology of inflammation existed, and consequently one obscure subject was being explained by another, almost equally obscure.

In 1844 Rokitansky opposed the inflammation theory and offered in its place the hypothesis, that material detrimental to the tissues of the arterial wall was deposited from the blood stream. Opposed to this view were Engel, Crisp, and Neumann, while Donders and Jansen were in support of the Vienna pathologist.

The important studies from this point on, as the microscope and histological technique improved, were concerned with the minute anatomy of the bloodvessels, also the physical laws governing their degree of dilatation, elasticity and resiliency.

Resse, a strong opponent of Rokitansky, presented important histological observations in support of the theory of *interrupted nutrition* and new connective-tissue formation.

It was Virchow, however, who was the strongest opponent, and to him is attributable the complete overthrow of the humero-pathological doctrine of Rokitansky. Virchow divided the process into two: first, a simple degeneration; second, an atheromatous degeneration. The first he considered as a passive occurrence, while the latter he looked upon as an active formative process. The hyperplasia of the intima he regarded as any other chronic inflammatory process. The sclerosis he considered to be a chronic inflammation, as had Dittrich before him, and to it he gave the name "endarteritis chronica deformans."

With the advance in the knowledge of the minute anatomy of the vessel wall, but more especially of the pathology of inflammation made in consequence of Cohnheim's and Metchnikoff's observations, new questions arose, such as, "What is the relation of the vasa vasora to the inflammatory area, and what part do the white corpuscles play in the process?" and "How do they reach the non-vascular intima directly, from the blood circulating in the lumen of the vessel or from the vasa vasora?" At or about this time much emphasis was laid upon high blood pressure as a result of continued contraction of the arterioles.

Traube was the chief supporter of this the "mechanical theory," while Rindfleisch suggested that disturbances of nutrition from slowing of the current were responsible for the disease of the wall. These were, however, only hypotheses. By careful histological search, in which they failed to find any means of communication between the non-vascular intima and vascular other coats, Götte, Koester, Stronganow, and Talma concluded that the intima became affected from the blood circulating in the lumen of the vessel. Koester, together with Friedländer, and before them Virchow, proved that cells do pass from the vasa vasora to the intima. Trompeter and Krafft, scholars of Koester, now made the following important contributions:

1. The media is always involved at or about the same time as the intima (mesarteritis).
2. That the vasa vasora play an essential part in the process of disease of the vessel wall.
3. They contributed also important anatomical facts.

Shortly after this the importance of the vasa vasora in their relation to the nutrition and disease of the vessel wall was shown by Durante's experiment.

These experiments showed that stopping the circulation of the blood in the lumen of the vessel was followed by no damage to the vessel wall, while cessation of blood flow in the vasa vasora was followed by destruction of the vessel wall.

Thus, while many isolated facts of great value had been contributed, yet no very clear knowledge of the etiology and sequence of events in this prevalent disease up to this time existed.

Thoma, as a result of his studies, presented an entirely new theory based largely upon histological and clinical observation.

Thoma's studies were on three conditions:

1. Vessels in the fœtus and newborn.
2. Vessels in the stump of an amputated limb.
3. Vessels showing disease.

In the first of these, the fœtus and newborn, he observed that the obliteration of the umbilical blood track (Nabelblutbalm) proceeds by way of a thickening of the intima.

In the second (the vessels in the amputated stump) he observed also that obliteration of the vessel took place by a thickening of the intima. He concluded that this thickening took place in order to compensate for the lessened amount of blood consequent upon the cessation of circulation through these vessels. These he called "Physiological Thickenings."

In the third, arteriosclerosis, he finds the same factors as in the physiological process, but appearing in different sequence. He distinguished two processes in the diffuse form of arteriosclerosis.

1. Primary.
2. Secondary.

In the primary he finds a yielding of the vessel from loss in elasticity with a consequent widening of the vessel and slowing of the blood current. Connective tissue develops in the subendothelial layers of the intima, a process which tends to restore the relation between the vessel and its contents. Slowing of the current may in itself disturb the relation between vessel and contents and so require a compensatory thickening of the intima.

With advancing years in an individual the slowing of the blood current is, *pari passu,* met by this compensatory thickening, and the relation between blood stream, vessel wall, cardiac potentiality and blood pressure is thus maintained. It is certainly a beautiful theory.

In the secondary variety the process begins in the small vessels. In arteriosclerosis nodosa the diseased areas are considered as localized points of elastic weakening, the compensatory process taking place at these points. The twisting of the vessel Thoma considered as points of diminished elasticity yielding to blood pressure. We, therefore, have the circumscribed or nodular form (of Councilman) and the diffuse form, for both of which we have the explanation that there is a primary weakening of the elastic coat with dilatation and compensatory thickening of the intima by development of connective tissue between the endothelium and underlying elastic tissue.

Many are the supporters of Thoma's theory, among these being von Schrötter, who, in *Nothnagel's System,* declares the explanation as final.

On the other hand, there are those who oppose the view, as Beneke and Pekelharing, for example, who differ from Thoma in considering a disproportion between pressure and tension and diminished resistance to be primary factors.

Marchand, too, considers that the many cases of circumscribed involvement of the intima with an almost normal media argue against a uniform compensatory intima thickening, and Bollinger asks, "Is not the mechanical theory too much considered?"[1]

R. F. Fuchs is perhaps the most active opponent of Thoma's theory. He considers that it is difficult to prove that an initial slowing of the blood current occurs and declares that it is unreliable to build a theory upon an unknown and undeterminable foundation.

The French writers, of whom Huchard is the most prominent, consider the process as one not confined to the vessels but a systematic condition manifesting itself in the vessel wall as in the kidney, the liver, and elsewhere.

CHART IV.

Theories. Nineteenth Century. (Not Chronological.)

I.	Inflammation theory.	Many observers.
	Pathology of Inflammation not clearly understood.	
II.	Humero-pathological theory, 1844.	Rokitansky.
III.	Pathology of inflammation. Better understood.	Cohnheim. Metchnikoff.
IV.	Mechanical theory	Traube.
V.	Interrupted nutrition theory.	Rindfleisch.
	Question 1. Nutrition of vessel wall?	
	" 2. Passage of leukocytes into vessel wall from lumen of vessel or from vasa vasora?	
	" 3. Nutrition of non-vascular intima?	
VI.	Passage of leukocytes from lumen hypothesis.	Götte, Koester, Stronganow, Talma.
VII.	Nutrition from vasa vasora *only*, demonstrated.	Durante.
VIII.	Passage of leukocytes from vasa vasora to non-vascular intima, demonstrated.	Virchow, Koester.
IX.	Compensatory process and molecular theory.	Thoma.

For further details see Prof. Jones' monograph on "Arteriosclerosis," to which I am indebted for much valuable information.

CHART V.

Nomenclature. Nineteenth Century. (Not Chronological.)

Arteriosclerosis	Lobstein.
Arteritis.	
Atheroma	Haller.
Arteriocapillary Fibrosis	Gull and Sutton.
Periarteritis.	
Periarteritis Nodosa, 1866	Kussmaul.
Obliterative Endarteritis, 1876	Friedländer.
Endarteritis, Chronica Deformans	Virchow.
Endarteritis Nodosa or Conscripta Diffusa	Councilman.
Mesarteritis	Trompeter-Krafft.
Angiosclerosis	Thoma.

Subdivisions of aneurysms not given, as these refer more especially to gross than to histological appearances.

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[1] Allbutt, vol. vii. p. 303.

CHART VI.

Epochs.

Prior to 400 B.C.	Complete ignorance.	
400 B.C. to 300 A.D.	Beginning of accurate observation.	ANATOMICAL FACTS.
300 to 1500	Decline of Anatomic study. Superstition and prejudice.	
1500 to 1600	Revival of ANATOMY. Anatomical Laboratories.	
1600 to 1700	PATHOLOGY. Pathology (Gross). Pathological Laboratories. SYMPTOMATOLOGY and Morbid Pathology.	
1700 to 1800	Methods of PHYSICAL EXAMINATION.	CLINICAL.
1800 to 1900	PATHOLOGICAL HISTOLOGY. BACTERIOLOGY. PHYSIOLOGICAL CHEMISTRY.	

Each of the above represents the dominant consideration of its time.

Such, then, is the history of the work done upon the arteries from the earliest epochs of medicine to our own times. It represents a vast expenditure of thought by the very ablest scientific thinkers, especially of the last three centuries.

There yet remain unanswered many questions, from among which the following have been selected, with the hope that some facts may be presented which may prove suggestive to other observers, stimulating them to investigate this widespread and important disease. The questions are:

1. Arterial disease in animals.
2. The earliest age in which arterial disease occurs in man, and at what age it is most common.
3. The teratological factor.
4. The relation of syphilis and pulmonary tuberculosis to aneurysm.
5. Relative frequency of aneurysm in the white to colored races.
6. Relation of arteriosclerosis to aneurysm.
7. The physical properties of the arterial wall.
8. The nature of the change in the elastic tissue, which is conceded to be the most important in arterial disease.

ARTERIAL DISEASE IN ANIMALS.

The determination of the presence or absence of arterial disease in animals presents many points of interest, such as the influence of strain, as in the beasts of burden; the influence of disease peculiar to man, as syphilis; the relative elasticity of the animal's vessel and that of man, etc.

The animal's vessel possesses a greater degree of elasticity, and this may enable the vessel to withstand strain for a longer time without yielding. This offers a fruitful field for work. Horses, and more especially asses, are subject to aneurysm of the superior mesenteric and aorta due to a parasite—the *Strongylus Armatus.*

Many authors state that peripheral aneurysm is practically unknown in domestic animals.

Pigs and dogs are stated to be occasionally the subjects of aortic aneurysm.

I examined the aortas of 103 domestic animals without finding any evidence, *in the gross*, of arterial disease.

In the aorta of the steer there occurs just beyond the first portion of the arch a thickening which is, however, not pathological. This is occasionally found in man.

EARLIEST AGE AT WHICH ARTERIAL DISEASE OCCURS IN MAN AND THAT AT WHICH IT IS COMMONEST.

It is generally stated that arterial disease in children is rare, but no definite age limit is given. Thus, Keating says: "Endarteritis with degenerative changes seems to be a rare affection in children; it would appear that the bloodvessels of the brain, and the walls of the aorta are the points chiefly affected, although other large vessels are occasionally the seat of atheroma." (*Cyclopedia of the Diseases of Children*, Keating, vol. ii. p. 874.)

Schmidt, however, reports a radial aneurysm in an infant eight weeks old; Syme, a popliteal aneurysm in a boy, aged seven years, and Hodgson an aneurysm of the carotid in a girl, aged ten years.

R. W. Parker says: "After careful search of the literature," he found only 15 cases of aneurysm in those under twenty years.

Through the courtesy of Dr. Mathias Nicoll, I was enabled to analyze 799 autopsies of children who had died at the New York Foundling Asylum, the ages varing as shown on this chart.

CHART VII.

Age Chart.

Analysis of 799 Cases. New York Foundling Asylum, 1898–1903.

Stillborn	to 3 months	85 cases.
3 months	to 6 "	140 "
6 months	to 9 "	155 "
9 months	to 1 year	140 "
1 year	to 2 years	141 "
2 years	to 3 "	87 "
3 years	to 4 "	37 "
4 years, 13 days		1 case each.
4 years, 1 month, 10 days		
4 years, 2 months, 4 "		
4 years, 7 months, 24 "		
4 years, 8 months, 12 "		
4 years, 9 months, 6 "		
5 years, 5 days.		
5 years, 14 "		
5 years, 3 months, 18 days		
6 years, 1 month, 13 "		
7 years, 4 months, 19 "		
10 years,		

Result of analysis:

Arteriosclerosis	1 case.
Endocarditis	1 "
Aneurysm	0 "

This list comprises tuberculosis in many localities. Pneumonia, empyema, gangrene of lung, scarlet fever, meningitis (all types), septicæmia, abscesses, nephritis, besides many other conditions which have not the bearing upon arterial disease that the above have. In addition to these there were 10 cases of congenital syphilis.

In all 799 cases there occurs only 1 case of atheroma and 1 case of endocarditis. No case of aneurysm.

This 1 case of atheroma was that of a boy, aged two years, six months and six days, who died of bronchopneumonia. There was an old appendicitis, focal necrosis in the liver and "slight atheroma of the aorta." Undoubtedly a case of sepsis.

In order to determine the age at which aneurysm is the commonest, 340 autopsy reports from literature were analyzed, with results as shown on this chart.

Chart VIII.

Aneurysm Cases from Literature.

1 to 5 years	2 cases.	2½ yrs. and 5 yrs
6 " 10 "	3 "	
11 " 20 "	27 "	
21 " 30 "	54 "	
31 " 40 "	102 "	
41 " 50 "	90 "	
51 " 60 "	43 "	
61 " 70 "	15 "	
71 " 80 "	3 "	
81 " 90 "	0 "	
91 " 100 "	0 "	
	340 "	

In regard to sex: of 347 autopsy analyses the following was found:

Aneurysm.	Male	280
	Female	67
	Total	347

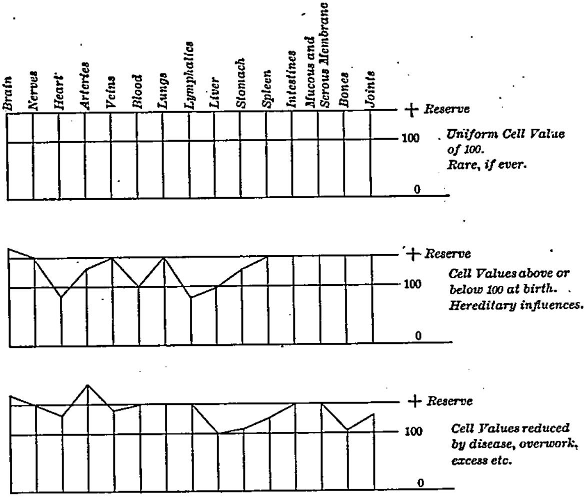
THE TERATOLOGICAL FACTOR.

A factor in arterial disease impossible to determine experimentally, but one none the less important, is that of teratology. The various parts of the body must be considered to possess a limited degree of reconstructive power.

Regeneration must have its limit. Cells of the kidney and liver, the heart, the brain, may not all have the same reconstructive energy. They differ from one another in an individual, and they differ from each other in different individuals. This is diagrammatically shown in Chart IX.

We might then speak of the "cell equation" of an individual, by which would be meant the perfection of the synergy (συν-ἔργον) of that system; thus the perfectly co-operative system would have a value of 100.

CHART IX.


Brain
Nerves
Heart
Arteries
Veins
Blood
Lungs
Lymphatics
Liver
Stomach
Spleen
Intestines
Mucous and Serous Membrane
Bones
Joints
+ Reserve
100
0
Uniform Cell Value of 100. Rare, if ever.
+ Reserve
100
0
Cell Values above or below 100 at birth. Hereditary influences.
+ Reserve
100
0
Cell Values reduced by disease, overwork, excess etc.

Teratological factor.

Individuals rarely, if ever, start with such a value. The demands during life too soon become greater on one organ than on another, so that this inequality becomes greater. A process of compensation is at work throughout the system struggling to approximate the perfect cell energy value of 100. In this work of compensation the vascular system has a great part. It, too, however, has its limitation.

SYPHILIS AND TUBERCULOSIS.

A question, the answer to which seems to be considerably confused, is what association is there between—

1. Aneurysm or arterial disease and syphilis; also what association there is between—

2. Aneurysm and pulmonary tuberculosis.

In the question of syphilis, for example, we find Schmidt places the figures at 29 per cent., Drummond at 100 per cent., while Gerhardt finds syphilis in 53 per cent. of the cases of aneurysm. Malmesten, on the other hand, says 80 per cent. Davidson, in the *Transactions of the Pathological Society of London,* states that in 114 autopsies he found 78 cases without syphilis, in 4 of which there was atheroma of the aorta; 28 cases with syphilis, 17 of which had atheroma—that is, 68 per cent. without syphilis, of which 5 per cent. had "atheroma," and 40 per cent. with syphilis, of which 60 per cent. had "atheroma." These are widely differing figures which probably arise from the great prevalence of syphilis. Had we a specific virus or organism of syphilis, as in the case of typhoid, cause and effect could be more certainly determined. I approached the question by analyzing 370 reports of autopsies on aneurysm cases.

In 106 of these syphilis was definitely mentioned. In 34 it was present; in 72 it was absent. This gives 32 per cent., or about that of Schmidt.

It would appear from these figures that syphilis is an important but by no means essential factor, nor is it invariably followed by disease of the vessel. In this question the age at which arterial disease is found must, of course, be taken into consideration.

In this connection Allbutt says: "I am beginning to regard atheroma of the aorta in young people as strong presumptive evidence of syphilis,"[1] though he regards syphilitic endarteritis as a distinctive process from atheroma from other causes.

I attempted to determine, if by present improved methods of treatment of syphilis, aneurysm was less common to-day than say fifty years ago, but reliable records on this point were difficult to obtain.

In regard to pulmonary tuberculosis, in 370 autopsies on aneurysm the lungs were examined and reported on in 98; 16 of these had tuberculous involvement of the lung, or 16 per cent.

Cases of pleural adhesions were not included in this estimate.

ARTERIAL DISEASE IN THE COLORED RACE.

Through the courtesy of Drs. Mathias Nicoll, L. Fougers Bishop, and B. T. Tilton, I was enabled to analyze 163 autopsy reports of the Lincoln Hospital, a hospital for the colored.[2] I was able to compare these, through the kindness of Dr. H. A. Christian, with 1928 autopsy reports of the Boston City Hospital, a hospital for the white. The result of these analyses is as follows:

Lincoln Hospital (colored).	Boston City Hospital (white).
Analysis of 163 autopsies.	1928 cases.
8 aneurysms, $4^8/_{10}$ per cent.	36 aneurysms, $1^8/_{10}$ per cent.
Thoracic, 6 Abdomen, 1 Iliac, 1 } 8	

Professors Osler and Welch gave me the opportunity to consult the clinical and pathological records of the Johns Hopkins Hospital. In the analysis of 1800 records the following data were obtained:

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[1] Allbutt, vol. vii. p. 309.

[2] While this hospital has recently admitted white patients to its wards, the records of autopsies on the colored patients only are included in the above.

Johns Hopkins Hospital.		Undetermined.	Total.
Colored wards	22 cases	7 cases.	52 cases.
White "	23 "		

Thus 52 aneurysms were found in 1800 autopsies. The number of white patients in this hospital outnumber the colored by about 4 to 1. These figures would indicate that aneurysm was found three or four times more frequently in the colored than in the white. Taking then the combined figures of these three large hospitals, the Lincoln, the Boston City, and the Johns Hopkins, we find aneurysm from three to four times as common in the colored as in the white.

Granting syphilis and exposure to be etiological factors, the greater frequency of aneurysm in the colored is to be expected. No reliable figures on the relative frequency of syphilis in the white and colored from which to draw conclusions on that point could be obtained.

ARTERIOSCLEROSIS AND ANEURYSM.

A question of great clinical value is whether general arteriosclerosis is or is not an indication of tendency to aneurysm.

This question suggested itself by the fact that cases of aneurysm of the aorta not infrequently have soft radials.

In pursuit of this point, 370 autopsy reports of aneurysm cases were analyzed, in 300 of which no reference was made to the peripheral vessels. In 70, however, the vessels were carefully examined throughout; 6 of these showed general arteriosclerosis; 64 of these showed no arteriosclerosis.

When the vasa vasora are involved first, localized softening of the media occurs with secondary changes in the intima. This weakening of the media leads to aneurysm. The intima in small aneurysms (sacculated aneurysm) we know is often not ruptured.

In arteriosclerosis, on the other hand, the intima is first involved, the media showing general loss of elasticity. General arteriosclerosis is not, therefore, necessarily associated with aneurysm.

An attempt was made to determine the relative frequency of arteriosclerosis and aneurysm in the various arteries.

Chart X. represents the result of this effort. In interpreting this chart it must be remembered that while multiple aneurysm is the exception, multiple arteriosclerosis (that is, several arteries involved at the same time) is common. Thus, of 100 sclerosed vessels, the radial will be found involved in 87 per cent., while of 100 aneurysms not one of the radial will be found.

It seems reasonable to conclude that in cases of generally thickened vessels, aneurysm is not a likely complication—*i. e.*, from the table here given it occurs in about 8 per cent.

DISTRIBUTION OF ARTERIOSCLEROSIS. The factors which determine the distribution of sclerosis of the vessels are varied. It may, however, be stated generally that when the peripheral vessels are markedly sclerosed the abdominal vessels are free from such involvement, and *vice versa*. Great functional demand upon any part is apt to lead to sclerosis of the vessels of that organ. Thus the cerebral vessels in brain-workers are often sclerosed. The coronaries in hypertrophied heart, the radials in laborers, etc.

CHART X.

Location Chart.

	Aneurysm (from Literature).	Arteriosclerosis (Thoma and Bregmann.)
Ulnar	½ per cent.	94 per cent.
Anterior tibial	¼ "	93 "
Subclavian	5 "	88 "
Cerebral { anterior, middle, posterior, ant. common, post. common, basilar, and mid. meningeal }	8 "	87 "
Internal carotid	1 "	87 "
Radial	0 "	86 "
Splenic	1 "	82 "
Popliteal	14 "	79 "
External carotid	¼ "	78 "
Axillary	1 "	71 "
Femoral	5 "	69 "
Common carotid	1 "	68 "
Ascending aorta	34 "	67 "
Abdominal aorta	8 "	64 "
External iliac	1 "	58 "
Brachial	2 "	55 "
Common iliac	1 "	
Innominate	3 "	
Thyroid axis	¼ "	
Pulmonary	3 "	
Coronary	1 "	
Cœliac axis	¼ "	
Sylvian	¼ "	
Basilic	¼ "	
Hepatic	2 "	
Plantar	½ "	
Profunda	¼ "	
Superior mesenteric	1 "	
Pons varolii	¼ "	

PHYSICAL PROPERTY OF ARTERIES.

What, now, are the properties of this wall whose chief element in construction is elastic tissue? The analogy between the elasticity of vessels and other elastic bodies at once suggests itself. It is found, however, by Wertheim and Weber, that the laws governing the elasticity of organized bodies, such as the vessel wall, differ from unorganized bodies, such as rubber. This difference is put down to the intrinsic moisture of organized bodies.

We have then to consider the organized structure apart from the unorganized, and must take into account four properties: elasticity, resiliency, extensibility, and resistance.

The elasticity of a substance refers (1) both to its ability to resist deformation and (2) to its ability after deformation to resume its original shape. These two ideas may be conveniently combined by supposing that no force is applied sufficient to permanently deform the object tested. Supposition number two above is, therefore, always understood to control the test; that is, that the object is not permanently deformed by the test.

The force at which permanent deformation occurs is the limit of the forces producing temporary only deformation, and this force is called the *elastic limit.*

For example, suppose that we stretch a wire or a rubber band a little, and then relax the stress. The elongation produced at first disappears when the stress is relaxed. We say that the recoil is equal to the elongation. Suppose that we then continue this process of alternately pulling and relaxing, measuring the elongations and the recoils, and increasing the pull every time. Up to a certain pull, or tensile stress, we find the recoils always equal to the preceding elongations; that is, that the deformation produced by the pull is not permanent.

After this limit (the elastic limit) has been passed we find the recoils are not as great as the preceding elongations, or that the object has stretched.

As a rule, the usefulness of anything that is permanently stretched is gone, therefore we study the properties of materials only below the elastic limit. These properties are the elastic properties.

The main elastic properties are:

1. The value of the elastic limit—*e. g.*, the pull in pounds per square inch that will permanently stretch the object. This has been defined above.

2. The rate at which the object stretches; that is, the extent of its elongation for a given pull. In a spiral spring this might be called the stiffness of the spring. Scientifically this is called the coefficient or modulus of the object's elasticity. This is a long and often confusing term, but the real idea it expresses is a very simple one. It is an application of the principle that "science is measurement." In other words, the coefficient is a numerical term by which the stiffness of objects may be compared; that is, of two objects stretching unequally under a given pull, the stiffest is that which stretches *least.* But if we measured the elasticity (that is, the coefficient of elasticity) by the stretch alone, the stiffest object would have the least numerical expression for its coefficient, which would be confusing.

For example: The spiral spring *A* under a pull of 100 pounds stretches one inch. The spiral spring *B* under a pull of 100 pounds stretches two inches. It is evident that *A* is the stiffer of the two springs, and that its coefficient is double that of *B*, yet its stretch is only one-half as much. So we say that the coefficient is measured by the pull divided by the stretch. Thus the coefficient of spring *A* is 100 pounds, and the coefficient of spring *B* is fifty pounds.

NOTE.—It is assumed that all other circumstances of the experiment are identical in the two objects *A* and *B*. It is apparent that while *A* is stiffer than *B*, yet it may be made, say of a different metal, so that *B* may have a higher elastic limit, although its coefficient may be lower.

Chart XI., Figs. 1 and 2, presents simple diagrams which will illustrate this.

Suppose that a pull of 100 pounds elongates the spring one-half inch with a recoil of the same extent. In the diagram Fig. 1 we illustrate this by the dot *a*. Similarly we find *b* and *c*. But when we make the pull 400 pounds we get the dot *d* with a recoil from *d* to the one-and-a-half-inch mark.

Let us join the points by a line as in Fig. 2. Then this line expresses all the elasticity characteristics of the object tested. The elastic limit is 300 pounds, for after this pull the extensions increase more rapidly than the recoils, or the spring has been permanently stretched—*e. g.*, two inches from 0 to 2. The back of the curve, like that of the resistance of the spring is broken at the "hump."

Chart XI., Figs. 3 and 4, shows another diagram containing the above figures, and the similar "strain diagram" of another spring.

In this case (Fig. 3), as in the hypothetical case, *A* is stiffer (or has a higher coefficient) than *B*, yet its elastic limit is only two-thirds that of *B*.

These diagrams represent the behavior of unorganized bodies. Let us now see how organized bodies behave under similar treatment.

As a rule, the strain diagram shows a straight line below the elastic limit; that is, for the portion of it from 0 to the "hump," so that the elasticity of the springs *A* and *B* may be compared by dividing the one pull—200 pounds which they have in common *below* the elastic limit—by the corresponding extensions, viz., one-half inch for *A* and one inch for *B*, making *A* twice as stiff (or elastic) as *B*, as before assumed. In these cases the coefficient for each spring is constant, though the different springs have different coefficients. Sometimes, however, the strain diagram is represented by a curve, as in Fig. 4. In this case we suppose that the curve is straight at the point in question as at *a*, *b*, *c*, or *d*. At these points draw tangents to the curve. We then compare the inclinations of the tangents. In this case the curve *C* shows that the stiffness is diminishing, and in curve *D* that it is increasing.

This latter increasing coefficient expresses the behavior of the artery as distinguished from the decreasing coefficient of unorganized bodies.

These diagrams are not fanciful, for the specimen may be made to write them autographically.

CHART XI.

FIG. 1.

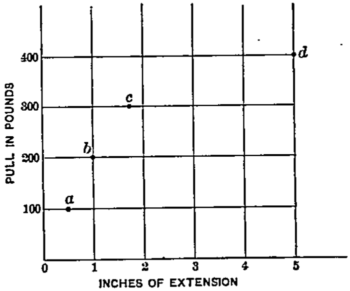


FIG. 2.

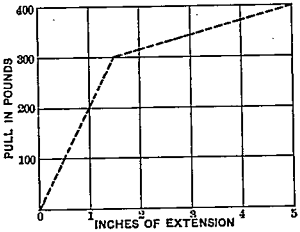


FIG. 3.

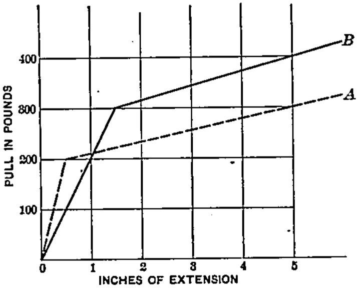
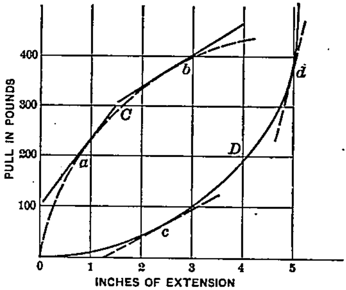


FIG. 4.



Coefficient of elasticity of organized as compared with unorganized bodies.

The elasticity of various organized substances is shown in Chart XII., also the relative elasticity of normal and diseased arteries.

CHART XII.

Relative elasticity of various tissues. (From Schäfer.)

Bones, dried	Similar to inorganic bodies.
Sinews, dried	Similar to fresh bone.
Nerves, dried	Similar to fresh sinew.
Muscles.	
Arteries.	

Relative elasticity of normal and diseased arteries:

Normal.	Weight, 100 grm.	Sclerosed.
Stretched.	Length.	Stretched.
16.5 cm.	7.5 cm.	9.9 cm.
Recovery:		Recovery:
8 cm.		8 cm.

Elasticity and resiliency diminished, especially elasticity.

To attain the greatest strength with a given amount of material, a cylinder should be constructed of layers, the elastic coefficient of which gradually increases from within outward. Now, this is exactly the arrangement of the different layers of the arterial wall; thus the greatest strength is attained by the smallest amount of material consistent with safety. Similar reasoning applies to the walls of a sphere, to which an aneurysm is comparable, from which it appears the main source of weakness in an aneurysm is, that the wall practically is composed of only one coat (Nicolls and Schäfer, p. 74). The human artery from this arrangement is physically perfect for the demands made upon it. This adaptability is demonstrated in Chart XI., Fig. 4, in which, according to Wertheim (as quoted by Schäfer, p. 72), the coefficient of elasticity of an excised strip of an artery is not constant but *with the extension increases.*

This property of increased coefficient of elasticity leads to a storing up of intermittent energy of the heart (Nicolls and Schäfer), which, if lost by disease, must greatly influence the rate of flow and thus furnish one of Thoma's essential factors in intima change.

In this connection, Dr. George McClellan calls my attention to an important anatomical fact, namely, the attachments of the great vessels to the deep cervical fascia, rendering them less movable, a fact which may be one reason why the arch of the aorta is so much more commonly affected than other arteries.

Very thorough investigation upon these physical properties of the arterial wall, together with histological observation upon the vessels experimented upon was carried out by August Luck in 1889, under the supervision of Prof. Thoma.

This work went to show that the normal artery has a greater resiliency than elasticity, while the reverse is true of the sclerosed vessel. In the initial stage of arteriosclerosis, that is, just before the thickening of the intima began, the vessel was found to be more expansile, but the elasticity was diminished. This change was found in vessels in which, histologically, no abnormal tissue could be detected.

CHART XIII.

Breaking strain of arteries.

(From various sources, together with personal observation.)

I. Normal vessels.	Pressures.
Carotid of goat.	2250 mm. Hg. 14 times the normal.
Carotid of dog.	3000 to 8500 mm. Hg.
Carotid of dog injured, intima and media slightly damaged.	At 450 mm. a pouch formed, but vessel did not rupture.
Human carotid.	1290 mm. Hg.
Large arteries rupture at lower tension than small arteries.	
II. Diseased.	
Localized sclerosis.	280 to 300 mm. Hg. Intima and media burst. Mercury trickled through adventitia.
Atheromatous plaques.	150 mm. Hg.
Generally thickened vessels.	1200 mm. Hg.

Breaking strain of veins.

Higher pressure required to rupture corresponding veins.

I am engaged in experiments along this line which seems a neglected but very important branch of the subject.

I am indebted to Captain Metcalfe, U. S. A., retired, and formerly instructor in ordnance and gunnery at West Point, for practical suggestions in the construction of apparatus to test the physical properties of arteries and for the explanation of the physical properties of unorganized substances.

HISTOLOGICAL PROPERTY OF ARTERIES.

All the evidence at hand goes to show that the seat of the most important change is the elastic tissue of the media. What is the nature of this change? It may be molecular, that is, purely physical; or it may be histological, that is, some tissue change. If the former, it can be detected only through changes in the physical properties of the artery and microscopic appearances characteristic of molecular change in the elastic contents. If the latter, that is, histological, we should be able to detect this through staining and chemical peculiarities. That the diseased artery shows marked variation from the normal in its physical properties, the facts just stated, together with evidence not here quoted, goes to show. The question then is, are there histological changes as well, or is it solely a molecular or physical change?

Attention was therefore directed to the microscopic appearance of the elastic tissue.

Thirty-three human arteries, consisting of the aorta, the popliteal, and coronary were subjected to stains selective of the elastic tissue. These vessels were both normal and diseased—the latter being composed of all stages of sclerosis to actual aneurysm formation. About 150 sections were thus treated and compared.

The stains employed were:

1. The Weigert.
2. The Unna Taenzer.
3. The Mallory.
4. Eosin hæmatoxylin stain was employed when information upon the vasa vasora was desired.

Each specimen was stained by all of these methods, so that they present a thorough demonstration of the relative merits of each method. A great variety of fixation agents was employed and experimented with. The best fixation agent is the Müller formalin, and the best stain the Weigert.

The steps in the staining are here briefly given.

TECHNIQUE EMPLOYED IN STAINING ELASTIC TISSUE. After experimenting with the various fixatives and hardening agents, as well as the different methods given in works on pathological technique for staining elastic fibres, it was found that the following gave best results:

I. *Weigert's Method* (Fig. 1). *Centralblatt für Allgemeine Path. u. Path. Anatomie,* May, 1898, vol. ix. p. 289.

Tissue stained equally well when fixed and hardened in Müller, formol or Müller-formol.

Formula is:—

Fuchsin (not Rubin S)	1 per cent.
Resorcin	2 "
Water	200 c.c.

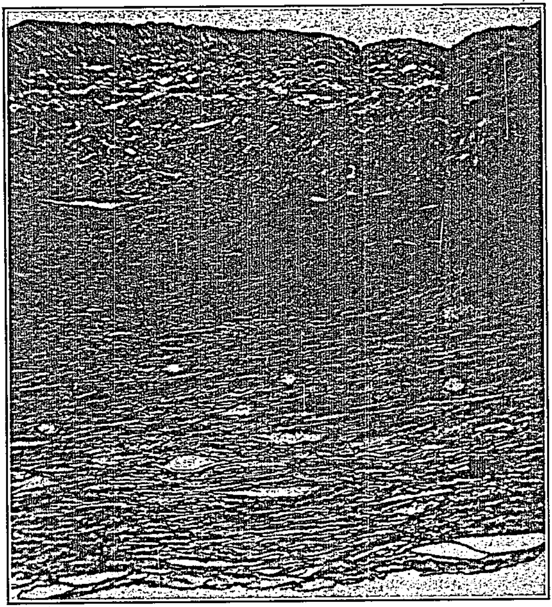
Boil in a porcelain capsule. When boiling add 25 c.c. liq. ferri sesquichlorati (Ph. G. III.), and boil 2 to 5 minutes longer, stirring meanwhile. Cool and filter.

Throw away filtrate. When precipitate is dry, place it in same capsule and add 200 c.c. 94 per cent. alcohol. Stir and let it boil, fishing out the pieces of paper. Then cool, filter, and to filtrate add enough 94 per cent. alcohol to make 200 c.c. Lastly add 4 c.c. HCl.

Stain may be used repeatedly.

For the present work, tissue was embedded in paraffin and sections cut 8 to 10μ. It was found that the best stains were obtained in ten to fifteen minutes instead of twenty minutes to one hour, as advised by Weigert, then washed in 95 per cent. alcohol, left for a few seconds in 100 per cent., cleared in xylol and mounted in balsam. If overstained, acidulated alcohol hastens removal of excess.

FIG. 1.



Selective elastic-tissue staining. × 160. Aorta (human). Weigert. See text, p. 865. Note that the darker area in the middle of the section is due to a folding of the tissue and not to any histological change. (From the Cornell University Photomicrographic Laboratory, N. Y.)

Elastic fibres are dark blue against a pale or colorless background.

II. *Unna Taenzer, modified* (Fig. 2).

Müller, formol, Müller-formol, alcohol, and bichloride may be used for this method.

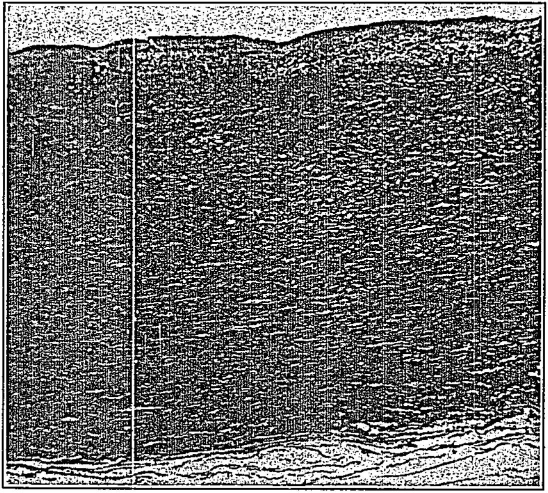
Formula for stain is:

Orcein (Grübler)	0.59 gm.
Absolute alcohol	40 c.c.
Distilled water	20 "
HCl	15 to 20 gtt.

Allow section to remain in this overnight, or about fifteen hours, then differentiate in:

Alcohol (95 per cent.)	20 c.c.
Distilled water	5 "
HCl	1 to 2 gtt.

FIG. 2.



Selective elastic-tissue staining. × 185. Unna Taenzer. See text, p. 866. (From the Cornell University Photomicrographic Laboratory, N. Y.)

Pass through absolute alcohol and mount in Canada balsam. Elastic fibres are brown against a very pale or colorless background.

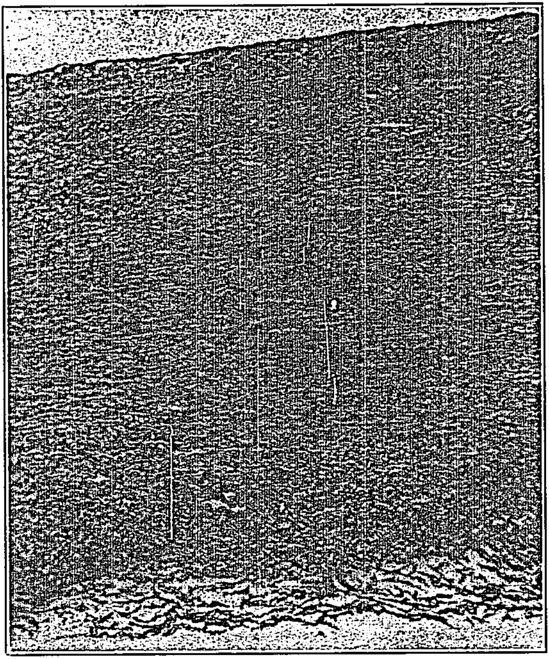
III. *Mallory* (Mallory and Wright's *Path. Technic,* p. 243, ed. 1897.)

Alcohol is the best hardening agent, a point emphasized by Mallory.

Phosphotungstic acid (1 per cent. aq. sol.)	100 c.c.
Hæmatoxylin	1 gm.

Sections are allowed to remain in stain overnight, washed in 95 per cent. alcohol, then 100 per cent., cleared in xylol and mounted in Canada balsam. If overstained acidulated alcohol will remove the excess. Fibres are a decided blue in a very pale pink background.

FIG. 3.



Tissue digestion experiments. Slightly sclerosed. × 160. Section of aorta (human) treated with 1 per cent. acetic acid. Stained with elastic-tissue selective stain. No change in tissue noted. See text, p. 867. (From the Cornell University Photomicrographic Laboratory, N. Y.)

The specimens, photomicrographs of which are here given (Figs. 1 and 2), show the following:

1. Vessels which in the gross display no signs of sclerotic change may, under the microscope, show slight thickening of the intima.

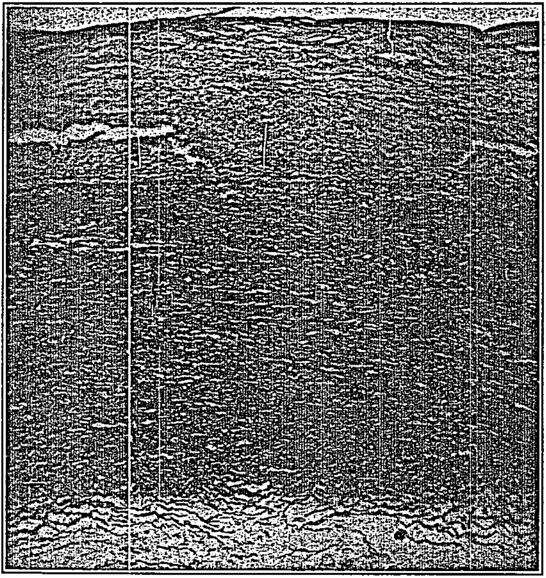
2. The elastic tissue shows the corkscrew character modified—the strands being straighter, more separated from one another, and in some instances fewer.

These then are probably mechanical changes, the stains failing to demonstrate any histological change in the elastic tissue.

DIGESTION EXPERIMENTS.

The next set of experiments was of a chemical nature, dealing with the reaction of the elastic tissue to digestants.

FIG. 4.



Tissue digestion experiments. × 185. More advanced sclerosis than preceding. Section of aorta (human) treated with 1 per cent. acetic acid. Stained with elastic-tissue selective stain. No change in tissue noted. See text, p. 866. (From the Cornell University Photomicrographic Laboratory, N. Y.)

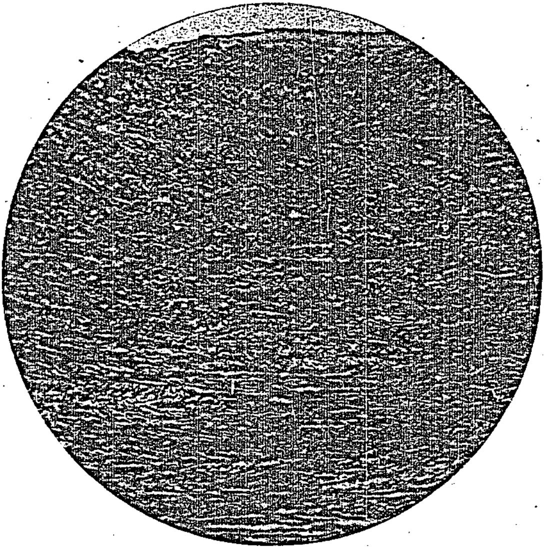
The object of this work was to demonstrate, if possible, through the action of digestants whether the change which takes place in the elastic tissue is of an histological or physical character.

The following experiments were therefore taken up. The digestants selected were acetic acid and potassium hydroxide.

Technique in the Attempt to Digest Elastic Tissue in Arteries with Acetic Acid and Potassium Hydroxide.

1. An aorta *hardened for several weeks* in Müller's fluid was chosen, and the area showing arteriosclerosis was subjected to the acid and alkali, as follows:

FIG. 5.



Tissue digestion experiments. × 185. Slightly sclerosed. Section of aorta (human) treated with 5 per cent. acetic acid. Stained with elastic-tissue selective stain. No change in tissue noted. See text, p. 866.

(*a*) *Acetic Acid.* 1. Strength of 1, 2, 3, 4, 5, 10 and 20 per cent. *acetic acid* were allowed to act in the incubator for twenty-four hours. Tissue being macroscopically unchanged, it was washed in running water for twenty-four hours.

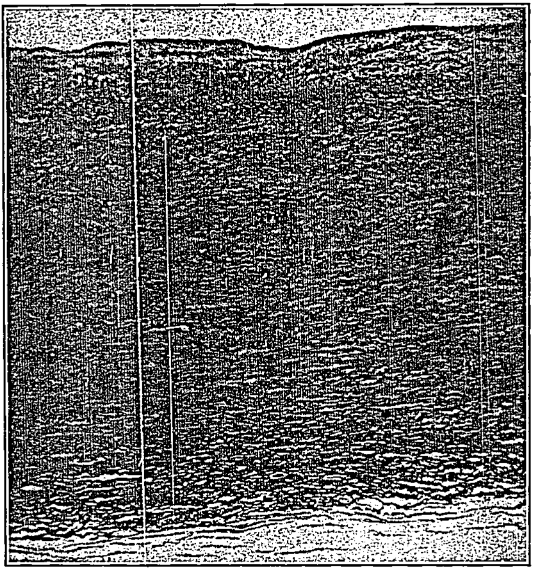
Dehydrated in

95 per cent. alcohol in incubator	24 hours.
100 " " " "	48 " (changed once).
Anilin oil " "	overnight.
Xylol " "	24 hours.
" paraffin " "	24 "
Soft " in oven	24 "
Hard " "	24 " and then embedded.

2. Specimens treated with same strength at room temperature for twenty-four hours appeared unaffected and were put through as preceding.

3. Another set of specimens were subjected to the same strengths of the acid for seventy-two hours at room temperature and no apparent change. Further treatment was the same as preceding. (Figs. 3, 4, 5, and 6.)

FIG. 6.



Tissue digestion experiments. × 185. More advanced sclerosis than preceding. Section of aorta (human) treated with 5 per cent. acetic acid. Stained with elastic-tissue selective stain. No change in tissue noted. See text, p. 870. (From the Cornell University Photomicrographic Laboratory, N. Y.)

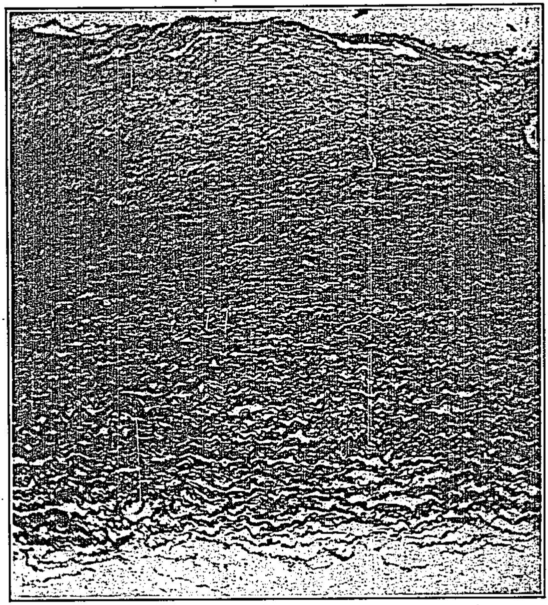
(*b*) *Potassium hydroxide*, KOH, 5, 10 and 20 per cent., in incubator for twenty-four hours, dissolved the tissue.

At room temperature for twenty-four hours, 10 and 20 per cent. partially disintegrated; 5 per cent. was left soft but intact; latter washed in running water for two hours.

Tissues were disintegrated. The strengths 5 per cent. and above are, therefore, not practical. (Figs. 7 and 8.)

Fresh material with marked arteriosclerosis was selected for another trial.

FIG. 7.



Tissue digestion experiments. × 185. Section of aorta (human) treated with 1 per cent. KOH. Stained with elastic-tissue selective stain. No change in tissue noted. See text, p. 871. (From the Cornell University Photomicrographic Laboratory, N. Y.)

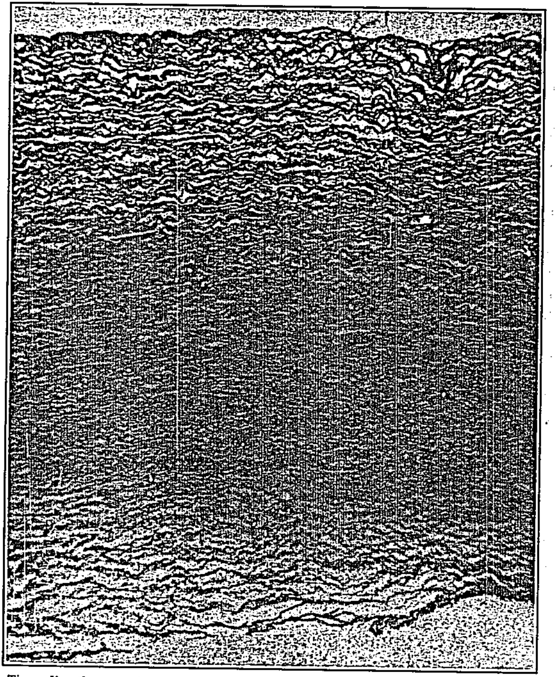
(*a*) 1. *Acetic acid*, 5, 10 and 20 per cent., in incubator twenty-four hours. Tissue softened.

80 per cent. alcohol, room temperature	24 hours.	
95 " " " "	24 "	
100 " " " "	24 "	
95 " " " "	24 "	
80 " " " "	24 "	
Running water	24 "	
80 per cent. alcohol in incubator	24 "	
95 " " " "	24 "	
100 " " " "	48 "	(changed once).
Anilin oil " "	24 "	
Xylol " "	24 "	
" paraffin " "	24 "	
Soft " in oven	24 "	
Hard " "	24 "	(embedded).

2. *Acetic acid*, 5, 10 and 20 per cent., at room temperature for twenty-four hours. Tissue quite firm and treated as above.

(*b*) *Potassium Hydroxide.* 1. KOH, 1, 2, 3, 4, 5, 10, 15 and 20 per cent. in incubator twenty-four hours; 5, 10 and 20 per cent. completely disintegrated; 4 per cent. partially broken up and fell to pieces in transferring to alcohol; 3 per cent. very soft but intact; 1 per cent. and 2 per cent. fairly firm. Last three put through same process as II. *a*.

FIG. 8.



Tissue digestion experiments. × 185. Section of aorta (human) treated with 2 per cent. KOH. Stained with elastic-tissue selective stain. No change in tissue noted. See text, p. 871. (From the Cornell University Photomicrographic Laboratory, N. Y.)

2. KOH, 1, 2, 3, 4, 5, 10, 15 and 20 per cent. at room temperature for twenty-four hours; 10 and 20 per cent. dissolved; 5, 4

and 3 per cent., very much softened, 2 and 1 per cent. less so, but carried through the different grades of alcohol and media as preceding.

CONCLUSION. The elastic tissue having resisted the digesting agent is probably not the seat of any histological change. It has probably undergone a molecular or physical change.

Selective stains and digesting agents failing to detect any histological change leave the molecular change theory while not proven yet with points in its favor. It leaves, however, the question of vasa vasora change untouched. To this source for early nutritive changes one inevitably comes, though it cannot in all cases be shown. This question is not given such slight reference with any disregard to its importance, but the search was on other lines, and the changes in the vascular supply to the vessels is mentioned here that it may not be thought to have been overlooked.

These experiments, as far as I know, have not before been attempted. Mall's experiments in digesting, with running water, all the substances of an organ leaving only the trabeculæ are of another nature. I regret that a greater number of specimens was not subjected to these experiments, but the specimens, photomicrographs of a few of which are here presented, required many months in the preparing, as the methods are long and tedious and the material could only be of the freshest, Museum specimens being out of the question.

The work is published not as a demonstration of any important conclusion, but merely as a suggestion which may yield better results to workers in the field of physiological chemistry.

CONCLUSION.

To sum up the findings then:

1. Arterial disease appears to be rare, almost unknown in animals. Syphilis being probably peculiar to man is by this observation placed more firmly in the list of etiological factors.

2. Arterial disease in children under six years, even in those who are victims of congenital syphilis, is practically unknown. In those from six to fifteen years it is rare. It is found in the initial stage most commonly between the ages of thirty and forty years.

3. The teratologic factor, though an undeterminable one, is of great importance.

4. Arterial disease seems to be attributable to syphilis in about 32 per cent.; to tuberculosis in about 16 per cent.

5. The facts presented go to show that the colored race is affected about four times more frequently than the white.

6. General arteriosclerosis seems to be not commonly found with aneurysm and its presence may be considered as evidence against the probable development of aneurysm.

7. Staining with selective stains and treating with a chemical which digests tissue shows the elastic tissue to be free of histological alterations, suggesting that this tissue undergoes physical or molecular rather than histological change.

To Dr. Jagle, who assisted me in the histological and digesting work, I want to express my appreciation of the patience and thoroughness displayed throughout, but especially at times discouraging to us both. Also to Dr. McClellan and Professor Ewing I am indebted for valuable suggestions. From Dr. Elser I obtained practical aid in the digestion experiments, and Dr. George Warren rendered me assistance in examination of autopsy records, without which much of the material here presented could not have been collected. To Dr. Schoenberg I am indebted for aid in collecting the foreign literature.

I wish to express my appreciation of the opportunity given me by the committee of the Mütter Museum to present this report, trusting that the observations may prove of value to the practitioner and offer suggestions to those who have the opportunity of the laboratory in which to work out these problems.

REPORT OF A CASE OF LAMINECTOMY FOR GUNSHOT WOUND OF THE SPINE.

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W. M. entered my service at the Samaritan Hospital August 11, 1903. He is a farmer by occupation, twenty-five years of age, white, born in the United States, and married.

He complains of severe pain at the lower part of the thorax, which extends around the body, and has some of the characteristics of a "girdle sensation." He also complains of nearly complete paralysis of his legs, especially of the left one.

The patient's father is alive and well, but his mother died of consumption. He has one sister who is alive and well, and one sister who died of la grippe.

W. M. says that he has never been ill or met with any accident excepting the one in question.

On June 14, 1903, he received a shot from a revolver just below and internal to the angle of the left scapula. The calibre of the bullet was 32. He immediately fell to the ground and could not move his legs at all for two weeks thereafter, and the bowel and bladder were also paralyzed. The wound bled but little and he at once experienced severe pain around the lower part of the thorax,